STUDY OF MORTALITY OF ONTARIO GOLD MINERS 1955–1977

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STUDY OF MORTALITY OF ONTARIO GOLD MINERS

1955-1977

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EXECUTIVE SUMMARY

In an earlier study a significantly increased lung cancer risk in gold miners as compared to an appropriately matched male population of Ontario and as compared to a cohort of nickel/copper miners was observed. This report deals with the possible causes of this increased risk.

The primary goal of this study was to determine if the observed higher lung cancer risk in gold miners was work related or if differences in life style factors could explain their higher risk.

A secondary aim of the study was to search for possible associations of lung cancer risk with specific contaminants in the mining environment.

An historical prospective cohort study with a nested case - control study was used in the investigation.

Conditions for inclusion in the gold miners' cohort were changed since the last report. Only men actually working in an Ontario gold mine at some time between January 1, 1955 and December 31, 1977, were now included in the study. Miners who no longer worked in a gold mine on January 1, 1955, but who chose to continue having miners' examinations after that date, were considered a self-selected group that

might attend miners' examinations for certain health reasons and therefore could introduce a bias in the further evaluation. This group of gold miners was now excluded from the cohort under study. This change resulted in both fewer observed and expected deaths from various causes but did not change appreciably the relative risk for these causes, with the notable exception of silico-tuberculosis, which declined from 41 observed deaths in the earlier study to 23 deaths in this report. Also the number of deaths assigned to silicosis declined, as a result of the change of the definition of gold miners, from 111 deaths reported earlier to 39 deaths.

As expected, cigarette smoke was by far the most important cause of lung cancer in gold miners. The relative risk of smoking as compared to non-smoking gold miners is a factor of 7. A comparison of smoking habits of gold miners and nickel/copper miners showed no significant differences that could possibly explain the greater lung cancer risk in gold miners as compared to nickel/copper miners. There are also no obvious differences in other life style or socio-economic factors that could explain the difference in lung cancer risk between the two cohorts.

The higher lung cancer risk in gold miners can also not be explained by age differences between gold miners and nickel-/copper miners. Gold miners show an increased lung cancer risk in all age groups that are normally affected by lung cancer. The greatest increase is, in fact, in the 55 to 64 year age group that is well represented in both gold and

nickel/copper miners.

Having failed to find that life style factors provide an acceptable explanation of the increased risk of lung cancer, associations with work related factors were sought.

The lung cancer risk in gold miners showed no association with the total number of years of exposure underground. The total number of years of work underground is not necessarily a valid surrogate for exposure when examining the relation between exposure and response. This is particularly so if there are great differences in exposure rate with time.

Improvements in ventilation and dust suppression techniques were carried out in the mines over the years. Major improvements occurred in the mid-1940's. The relation of years worked underground prior to 1945 and lung cancer risk was therefore investigated. The lung cancer risk increased significantly with the number of years of dust exposure in the mines prior to 1945. Exposure after 1945 contributed very little to risk.

An association between year of start of gold mining and lung cancer risk was demonstrated. Men who started gold mining in the early years under less favorable conditions show a higher risk than those who started mining later.

The results of the case-control study indicate an increasing lung cancer risk with an increasing number of months worked in high dust exposure.

All the findings of the study indicated above point to a significant occupational contribution to lung cancer risk in gold miners.

A secondary aim of the study was to investigate possible associations of lung cancer risk with exposure to specific environmental contaminants in the gold mines. Among the factors investigated are exposure to high dust levels, silicosis and exposure to silica, arsenic, fibrous materials in the ore and radioactivity.

Silicosis became a primary suspect, as there is some experimental evidence that inhaled silica can, under certain conditions, give rise to lung cancer in rats. Also some epidemiological evidence pointed in that direction.

The data in this study indicate a significantly increased lung cancer risk in each sub-cohort of non-silicotic and silicotic miners. The relative risk of dying of lung cancer, however, increases with increasing X-Ray rating from non-silicotic to silicotic (sub-cohort A to C).

When comparing smoking habits of silicotics with those of non-silicotics, it was found that silicotics had a history of smoking more than non-silicotics. This finding supports a hypothesis that smoking increases the risk of silicosis in an environment where silicosis does occur and so also indirectly affects lung cancer risk in silicotics.

The association of smoking and silicosis rating with lung

cancer risk was investigated using a multivariate analysis. Both smoking habits and silicosis rating separately showed a significant association with lung cancer risk. The association of X-Ray rating with lung cancer risk could no longer be demonstrated when smoking was included in the model.

These findings support the hypothesis that smoking increases both the lung cancer and silicosis risks and that the observed increased lung cancer risk in silicotics as compared to non-silicotics is at least partly the result of differences in smoking habits between the sub-cohorts.

Silicosis in Ontario gold miners can also be viewed as an indicator of high dust exposure and of greater exposure to cancer-causing agents present in the mine environment. The association of silicosis with increased lung cancer risk in gold miners is therefore likely to be spurious.

Information on the geology of the various gold mines was obtained from the Geological Survey of Ontario. Based on the information, mines were categorized into groups according to:

High or low arsenic levels
High or low fibrous materials
High or low sulfide levels
High or low radioactivity
High or low silica levels.

Various relative risk models were used to investigate the association of lung cancer risk with the above parameters in combination with high dust exposure and smoking information. The application of these models to the available data on Ontario gold miners suggests a positive association of lung cancer risk with exposure to high dust levels in combination with high levels of arsenic and/or fibrous materials. No association could be demonstrated between lung cancer risk and exposure to silica or elevated radon levels predicted from the geology. There is, therefore, a suspicion that both high arsenic levels and high levels of fibrous material in the ore are associated with increased lung cancer risk. There also is an indication that high dust levels in combination with high levels of arsenic or fibers in the ore are not the only causative agents for lung cancer in the mine environment. The data obtained from the cohort study indicate that miners who never worked in high dust levels combined with high arsenic or fiber levels still show a significantly increased lung cancer risk. Other factors in the mine environment, such as radon and its short lived progeny, might well be responsible for this part of the risk.

It should, however, be noted that geology is not a good indicator of elevated levels of the progeny of radon in the mine atmosphere. The more direct way to look for an association between exposure to radon and its progeny in the mine air and lung cancer risk could not be used. Levels

of radon daughters in Ontario gold mines in the past are not known and the results of recent measurements are available only in some of the mines where members of the cohort worked.

The possible effect of aluminum prevention of silicosis on lung cancer risk was investigated. Out of a total of 7057 members of the gold miners' cohort only 327 men (5 percent) never indicated exposure to aluminum prevention. Both exposed and non-exposed groups show a significantly increased lung cancer risk. There is, however, no significant difference in risk between the two groups. Men who started gold mining in Ontario before 1936 or some ten years prior to the introduction of aluminum prevention into Ontario gold mines have a higher lung cancer risk than those who started later. The case-control study does not indicate an association of years exposed to aluminum prevention and lung cancer risk. For all these reasons a hypothesis that aluminum prevention increased lung cancer risk in Ontario gold miners failed to find support in the data.

A comparison of the classification of lung cancers by cell type carried out in hospitals and their reclassification by Dr. Ritchie showed major discrepancies. These will be discussed in a separate publication. For the purpose of this study, only the results of the reclassification were considered. There were no major differences in the distribution of lung cancers by cell type between gold miners and nickel /copper miners.

No causative agent for the increased stomach cancer rate in this cohort was identified. The high proportion of immigrants in the cohort was found not to be related to the elevated rates.

INTRODUCTION

In an earlier study (Mu83) significantly more deaths from certain causes were observed in a cohort of Ontario gold miners than expected, based on an appropriately matched male population of Ontario. These causes included respiratory tuberculosis, cancer of the stomach, cancer of the trachea, bronchus and lung, non-malignant diseases of the respiratory tract and violent deaths. In contrast, only more violent deaths than expected were found in nickel/copper miners and no increase of death rates due to any type of cancer or non-malignant disease of the respiratory tract could be detected in this group.

Other authors have not demonstrated an increased lung cancer risk in hard rock miners generally and specifically in gold miners (Br86). The geology, as well as work practices and temporal factors, might well differ from mine to mine and it is therefore not surprising to find contradictory results in various studies of gold mining populations.

This part of the study deals with cancer of the trachea, bronchus and lung. Specifically it attempts to investigate possible reasons for the observed increase in risk of dying of this cause in the underground gold miners' cohort, a risk not observed in underground nickel/copper miners.

The primary objective of the study is to determine if the observed lung cancer risk in gold miners is life style related or if conditions in the working environment

significantly contributed to the observed risk.

A secondary objective of the study is to investigate possible associations between exposure to specific pollutants in the underground mine environment and lung cancer risk.

Several reasons for the increased lung cancer risk in gold miners were considered:

a) The male population of Ontario might not have been an appropriate control group.

b) Ontario gold miners might possibly smoke more than the male population of Ontario and nickel/copper miners.

c) The increased risk might be related to some environmental factors in gold mines.

METHODS

The Study Population :

In an earlier study, "non-uranium miners" were defined as men with 60 or more months of mining experience including one-half month or more of mining experience in an Ontario mine, as long as they did not start work in an uranium mine. An additional condition was that they were known to have been alive at some time between January 1, 1955 and December 31, 1977, as indicated by attending the yearly miners' examination. (For details see Mu80,83,84.)

A gold miner was defined as a "non-uranium miner" who has spent 85 percent or more of his total mining experience, in and outside Ontario, in Ontario gold mines.

Miners were further classified as full-time underground miners if they spent more than 50 percent of the total time of dust exposure in Ontario as underground miners. Otherwise they were considered part-time underground miners. In this context time spent in non-dusty surface jobs was neglected. Once classified as full-time underground miners, jobs in dust exposure on the surface and underground were no longer differentiated and the terms "work in dust exposure" and "underground work" have, for the purpose of this report, the same meaning.

When examining the actual data in greater detail, it was found that some gold miners continued to attend miners' examinations when they no longer worked as active miners. It was suspected that those who attended the miners' examinations after stopping work as miners might be a selected group of men with some medical problem, notably early silicotic changes. As a result, some men, who had a miners' examination in or after 1955 when they no longer worked as miners, would not have been included in the study population if they had not volunteered for further miners' examinations. These men are now excluded from the study population.

As a result of these changes in the definition of full-time underground gold miners the number of these men is now 7057. They were followed up from the time of entering the study to the end of 1977, to entry into uranium mines or to death, whichever came first. They were followed for a total of

111,587 person-years. The number of lung cancer deaths observed in this cohort is 165 for the years 1955 to 1977 and the number of expected lung cancer deaths is 117.6 (SMR=140, P=6x10-6).

The definition of the full-time underground nickel/copper miner was changed in a similar fashion as the definition of a gold miner. In nickel/copper miners the change in definition had, however, no marked effect on mortality as compared to the previous report (Mu83).

As in the earlier reported study, men with known asbestos exposure or men who have also worked in a uranium mine outside the Province of Ontario or in a uranium processing plant were excluded from these cohorts.

The Death Search :

A nominal roll of the study cohorts was linked by machine with the Canadian Mortality Data Base over the years 1955 through 1977. Surname, full given names, year, month and day, as well as place of birth were used as personal identifiers. Whenever available, the Social Insurance Number was used as well, in order to determine the mother's maiden name, which is also available on the death certificate. A minimum probability for acceptability of a positive link was chosen and manual resolution of positive links was carried out making use of further information, which is not available on the computerized Canadian Mortality Data Base. The efficiency

of the death search was tested on a sample of 1,097 known deaths in nickel/copper miners. The record linkage procedure failed to discover 69 deaths, or 6 percent of the total. (For further details on the death search see (Mu83))

Types of Study Carried Out :

a) The Cohort Study :

Person-years at risk were calculated from the time of entering the study to the end of 1977, to death or to the time the man became a uranium miner, whichever came first. A man entered the study at the first mining examination between 1955 and 1977, when he fulfilled the required conditions for inclusion into the study as "non uranium miner". The year of entry into the study and the year of leaving the study was counted as half a year. (For further details see (Mu83)).

Two control populations were used to calculate the number of expected deaths by cause group; the male population of Ontario matched by five-year age groups and five-year calendar periods, and the cohort of Ontario nickel/copper miners matched by age groups. Broader cause groups had to be used for the calculation of "expected" cases based on the cohort of nickel/copper miners because of the relatively small population size.

Members of the gold miners' cohort were subdivided into three sub-groups by calendar year of starting gold mining in Ontario. Sub-group 1 started gold mining in Ontario prior to

1936. Sub-group 2 started between 1936 and 1944. Sub-group 3 started in 1945 or later.

Members of the cohort were further subdivided into 3 sub-cohorts by their X-Ray rating into sub-cohort :

A: Ontario X-Ray code less than 4, ILO code 0/0.
B: Ontario X-Ray code 4, ILO code 0/1 or 1/0.
C: Ontario X-Ray code greater than 4, ILO code 1/1 or greater.

Each man contributed person-years at risk and expected lung cancer deaths to individual sub-cohorts as he moved from a lower to a higher X-Ray code. A miner could enter the study in sub-cohort A and contribute person-years and expected lung cancer deaths to this sub-cohort up to the time he moved into sub-cohort B. Person-years at risk and expected deaths were apportioned in a similar way to sub-cohorts B and C.

b) The Case-Control Study :

The lung cancer risk of a gold miner might well depend on his smoking habits as well as his exposure to various environmental pollutants in the mine. The exposure of an underground gold miner to environmental pollutants in the mine depends on a number of factors such as years of exposure; time period of exposure; the geology of the various mines; the dust concentration in the mines, that were changing with time period; the presence and concentrations of other contaminants in the mine environment; the occupation of the man in various periods; and a number of other parameters that are not easily ascertained. Not all of these parameters could be studied in the total cohort of 7057 men. A

combination of the cohort approach and a case-control approach was therefore used.

Each man who died from lung cancer was matched randomly to a control, who was a gold miner, never became a uranium miner, and was born in the same year as the case. Every possible effort was made to trace the men or their next of kin in order to obtain smoking histories for both cases and controls. Difficulties were encountered, however, in obtaining smoking histories on all the controls. Some re-matching has become necessary using controls from a 3.8 percent random sample of the gold miners cohort born before 1938. As a result, 108 pairs could be matched by year of birth exactly; seven pairs within one year; two pairs within two years; one pair within three years; and one pair within four years. In each of the pairs the control survived the cancer case. Only cases and controls with known smoking histories were included in the case-control study. As a result, 119 pairs out of a total of 165 pairs were included in the case-control study.

The Collection of Smoking Information :

Smoking information on men deceased from lung cancer was obtained whenever possible from next of kin. If next of kin could not be traced, hospital records were utilized. Information on year of starting and quitting smoking, as well as on amount smoked, was sought.

Smoking information was also sought on two control groups of miners: 1.) the control group of gold miners, used in the case-control study, and matched with the lung cancer cases on a one-to-one basis; and 2.) a random sample of 347 members of the gold miners' cohort born before 1938. Smoking information was obtained on 235 men.

A control group of 235 gold miners with smoking histories was thus assembled in addition to the 119 cases and controls with smoking histories used in the case-control study, as well as a comparison group of 330 nickel/copper miners with smoking histories.

In order to investigate the extent to which the 119 lung cancer cases are a representative sample of the total of 165 observed cases, certain comparisons were made. Tables A to E in the appendix give a description of the sample used in the case-control analysis in comparison with all lung cancer cases found in gold miners. The minor differences in the representation of men by year of birth, year of start of gold mining, as well as by other parameters indicated in the appendix Tables A to E in the case-control study, were unavoidable and were unlikely to produce any false associations between exposure and risk in the gold miners. It is, however, possible that some associations might have been missed as a result of it.

Work History :

Making use of the statements of the men at the yearly miners' examinations, a detailed work history was assembled for each man. The work history contains data on the mine in which the man worked during each calendar year, the number of months worked in each mine, and information on the occupation of the man in the mine during each year. The men indicated in the course of years more than 200 different occupations in the mines. Frequently identical occupations were named differently by custom at various mines at various time periods. With the help of mining engineers, the unions and individual experienced miners, this great number of jobs was categorized into eight occupational groups that were likely to differ in the degree of exposure to mine dust and blasting fumes at various periods.

The Classification of Exposure Rates :

Dust counts by konimeter were carried out in Ontario gold mines from the mid-1920s up to the end of the study period. The data are scantier the further back in time they go. Measurements were, however, carried out in relation to such typical occupations as raise drilling, drift drilling, stope drilling, tramming, work at crushers, etc. at different times. Nevertheless, only crude estimates can be made of exposure to mine dust of workers in the various occupational groups, by mine and calendar year.

Taking into account the small number of samples taken, particularly in the 1920's and 1930's, when some of the men started gold mining in Ontario, it was decided not to use actual numerical values of dust concentrations to which a miner was exposed in a given year. Instead the counts were grouped into high and low, taking into account the time period of exposure, camp and occupation of the man. High dust counts represent levels in the range of 500 to over 1000 particles, and low dust counts about 200 particles per cubic centimeter of air. Exposure of the men to mine dust was expressed in terms of high and low "dust-months".

The free silica content of the ore varies in the different mining camps from a low of 16 percent in Kirkland Lake to a high of 31 percent in the Fort William mining camp. The exposure to free silica was expressed as "high" in the Fort William camp and as "low" in the Kirkland Lake and Timmins mining camps.

Individual miners worked in high or low dust levels often in the same mine and period dependent on the type of job held by the man. It should however be recognized that both "high" or "low" "dust months" or "high" or "low" "silica months" are only crude estimates, and do not represent highly accurate exposure data. For this reason, only the crude classification into high and low dust levels was used.

No measurements of blasting fumes in the mine air were carried out. The organization of the blasting operations

has, however, changed quite substantially over the years resulting in diminishing exposures of the miners.

In the period prior to 1927 ventilation was poor in the gold mines and blasting operations were carried out at any time during the work shift. As a result, all underground gold miners had high exposures to blasting fumes during this period.

Between 1927 and 1939 blasting was still done during the shift, but men working in stopes and raises and also men engaged in tramming operations left the mine after the blasting operation was concluded. Men working in drifts returned and resumed work after allowing about half an hour for the fumes to dissipate. The exposure of drift workers was therefore greater than that of stope, raise and tramming workers.

During the period 1940 to 1947, drift workers no longer returned to work after blasting. Ventilation had also improved markedly.

Further improvement of the mine ventilation, as well as an improved organization of blasting procedures, limited to the end of the shift only, resulted in a further reduction of exposure of miners to blasting fumes. These improvements were introduced in the mid-1940s. The changes were gradual, occurring in most mines at or about the same period, but not exactly at the same time. The accuracy of the data for exposure to blasting fumes cannot be supported by any

historic measurements in the mine atmosphere and the degree of the exposure rate can only be inferred from information on the organization of blasting procedures and from changes in the ventilation of the mines. Exposure rates by calendar period and job classification have, therefore, been used only in qualitative terms. Table F in the appendix summarizes information on exposure levels to blasting fumes by calendar period and job classification used in the study.

The Ontario Ministry of Natural Resources provided, through its Geological Survey, data on the geology of the various mines. The varied geology was coded into groups indicating the content of:

a) arsenic as high or low.
b) sulfides as high or low.
c) fibrous materials present or absent.
d) elevated radioactivity present or absent.

Months of dust exposure were calculated for both the case and his matched control from beginning of gold mining in Ontario up to ten years prior to the death of the case or up to the time a case or control became a member of sub-cohort B. This was done in order to correct for the likelihood that the case stopped working underground long before death because of his illness or because of his X-Ray classification. The number of months worked in high or low silica, arsenic, fibrous materials, etc. were calculated in an identical manner.

The Classification of Lung Cancer Cases by Cell Type :

An effort was made to obtain information on the cell type of lung cancer cases from hospitals that performed either biopsies or post mortem examinations on members of the study population. In addition, tissue blocks, or, if not available, slides prepared in the hospitals where the men received treatment, were collected. Up to the time of writing, 372 histology reports were obtained from hospitals, including 85 reports on deceased gold miners and 41 on deceased nickel/copper miners.

Tissue blocks or slides from 321 miners who were members of the study population were obtained and re-evaluated as to cell type. These include 81 blocks or slides from gold miners and 37 blocks or slides from nickel/copper miners. Tissue blocks or slides were assigned code numbers. Therefore, at the time of re-evaluation of the individual case, neither the ore the man had been mining nor the histological findings in the hospital were known to the pathologist.* Only after the histological report was received was the code number linked to an individual and his appropriate cohort.

* Prof. A.C. Ritchie

RESULTS

The number of observed and expected deaths by cause over the period of follow up of the gold miners' cohort (1955 to 1977) is given in Table 1. The expected number was calculated from death rates of the male population of Ontario, matched for age and calendar period. The values of the Standardized Mortality Ratios (SMR) were calculated and the hypothesis, that the observed number of deaths by cause does not significantly exceed the expected number, was tested. The P-values are indicated only if less than 0.05. The one sided test was used, as the healthy worker effect reduces the number of observed cases in a variety of causes of death.

It will be noted that there are fewer deaths, both observed and expected, than indicated in Table 10 of the 1983 report of the Ontario Miners Mortality Study. This is due to the change in the definition of the underground gold miners' cohort, indicated earlier. There are, however, no significant changes in the SMR values compared to the earlier report except for silicosis and silico-tuberculosis.

The male population of Ontario is not necessarily an adequate control population for underground gold miners. Underground nickel/copper miners were, therefore, used as an alternative. The limited size of this population results, however, in larger errors in the estimate of expected death rates. Table 2 gives a summary of results of this comparison.

Smoking Habits :

One of the possible reasons for the observed increased lung cancer risk in underground gold miners could have been a difference in smoking habits between gold miners and nickel/copper miners or the Ontario population. Smoking habits were investigated in gold miners and nickel/copper miners using identical techniques described earlier. A comparison of smoking habits of nickel/copper miners and gold miners was therefore made. Table 3 summarizes the results of the comparison. No appreciable difference in smoking habits of the two cohorts could be detected.

Total Number of Years Worked Underground :

The total number of years worked underground in Ontario gold mines might possibly be related to lung cancer risk. In order to test this hypothesis, the lung cancer risk in sub-groups of gold miners, varying as to the number of years of underground exposure, was compared. In this calculation men moved from one "years worked group" to the next as they continued mining underground during the period of observation. Person-years were assigned accordingly. The results are summarized in Table 4.

Year of Start of Underground Gold Mining and Years Worked Underground Prior to 1945 :

If the increased lung cancer risk in underground gold miners

was linked in some way to factors in the mine environment, men who started mining in the earlier years, when dust suppression and ventilation were generally less effective, should be at greater risk of dying of lung cancer than those who started gold mining in later years. In addition, years of dust exposure in the early years, prior to major improvements in the underground mining environment, should be associated with an elevated lung cancer risk. Tables 5 and 6 demonstrate these points.

Table 5 gives, by calendar year of start of gold mining in Ontario, the number of observed and expected lung cancer deaths, as well as values for relative risk and probabilities that any difference between the number of expected and observed lung cancer deaths occurred by chance only. Significant differences between observed and expected lung cancer deaths were found both in men under the age of 65 years and in men 65 years and older, who started underground gold mining prior to 1936. In the age group younger than 65 years, the difference between expected and observed lung cancer deaths is still borderline significant in men who started underground gold mining between 1936 and 1944, but not so in men 65 years and older. No significant increase in lung cancer risk could be observed, for either age group, in men who started underground gold mining in Ontario after 1944. The relative risk of dying of lung cancer decreases with later start of underground gold mining of the sub-groups.

Table 6 indicates lung cancer risk by the number of years of underground employment in gold mines prior to 1945. In contrast to Table 4, which shows no increase in lung cancer risk with the total number of years of dust exposure in gold mines, Table 6 indicates an increasing relative risk with the number of years of underground dust exposure in gold mines prior to 1945.

The effects of year of first gold mining and years of dust exposure before 1945 were considered by using a log-linear model, which takes into account the effects of attained age and calendar period before evaluating the parameters of interest.

The relative risk of lung cancer declined with more recent entry into the gold mines (X^2 = 7.74, P = 0.005) and increased with increasing years of dust exposure before 1945 (X^2 = 8.33, P= 0.004).

X-Ray Rating and Lung Cancer Risk :

Table 7 indicates a significantly increased relative lung cancer risk in each sub-cohort by X-Ray rating. Using a log-linear relative risk function it was demonstrated that the relative risk of dying of lung cancer increases with increasing X-Ray rating of sub-cohorts from A to C (X^2 = 8.08, P= 0.005).

Tables 8 and 9 give results of the cohort study relating to both sub-group by start of mining gold in Ontario and sub-

cohort by X-Ray rating. The data indicate that the lung cancer risk decreases with later period of starting underground work in Ontario gold mines, and rises with the increasing X-Ray rating of sub-cohorts from sub-cohort A to C.

As Table 10 indicates, the increased lung cancer risk is not limited to one age group. All age groups where lung cancer deaths normally occur show a significant or borderline significant increase in lung cancer risk. The 55 to 64 years age group appears to show a higher risk than the 65 years plus age group.

Results of the Case-Control Study :

As indicated earlier, in the section on Methods, exposure of both case and control was calculated up to 10 years prior to the death of the case, or until either member of the pair became a member of sub-cohort B or C. The ten-year period of discounting exposure was used in order to correct for the inability of the lung cancer case to work in the mine, at times over several years, prior to death from lung cancer. It also became apparent, that on entering sub-cohort B or C, miners tended to move from underground jobs to surface jobs.

Smoking information could not be obtained on all the cases and the matched controls. Smoking habits were however considered essential in any further analysis of the data. It was therefore decided to utilize in the case-control study only

pairs with known smoking histories of both members. This reduced the number of pairs to 119.

A number of simple preliminary tabulations were carried out before embarking on more sophisticated analytical procedures in order to obtain a better understanding of the raw data. The results of some of these observations are demonstrated.

Table 11 compares the smoking habits of cases with their matched controls. For the purpose of this table, cases and matched controls who were smokers within 10 years prior to the death of the case are considered to be smokers. All other cases or controls were considered to be non-smokers.

The relative risk of smoking is 7 ($P << 0.001$).

There is no striking difference between cases and controls in the total number of years worked underground up to ten years prior to the death of the case or up to the time either case or control entered sub-cohort B. Cases worked an average 17.1 years underground, as compared to an average of 17.6 years worked underground by controls. The results of the cohort study indicate, however, that increased lung cancer risk is associated with years of dust exposure underground prior to 1945. Cases have more years of exposure in high dust levels than controls. Thirty-two cases worked a total of 211.8 years (average 6.6 years) in high dust levels, while 33 controls worked a total of 132.6 years (average 4 years) in high dust levels. These data give a preliminary indication that high dust levels might be

associated with an increase in lung cancer risk.

Table 12 compares the total number of years worked, the number of men and the mean number of years worked per man in high dust levels associated with a high content of various pollutants in the mine ore, in both lung cancer cases and their matched controls.

Table 13 compares cases and controls as to their X-Ray rating. The relative risk for a case to be a member of sub-cohort B or C compared to controls is 30/14 = 2.1. This result is statistically significantly different from a ratio of 1 (P = 0.02).

Table 14 gives the relative risk of X-Ray rating when both case and control are current smokers. If both case and control are current smokers, the relative risk of having an Ontario X-Ray code 4 or greater or an ILO code greater than 0/0 is no longer statistically significant.

Table 15 gives the number of both cases and controls who worked in various categories of exposure to blasting fumes, the average number of months worked in these categories of exposure and the total number of months. The table does not indicate any significant difference in exposure to blasting fumes between cases and controls.

Following the preliminary analysis of the data in the case-control study, the method of the matched case-control study was used (Th83) to analyze simultaneously the effect of

several types of exposure on lung cancer risk.

The most powerful environmental factor in relation to lung cancer risk was shown to be smoking. Models used therefore include, in all cases, smoking as one of the parameters in the analysis. The effect of smoking was modelled as current smoker if the man smoked within the last ten years before death of the case. Otherwise the man was considered a non current smoker. Table 16 summarizes the results of this analysis.

Table 17 gives the results of a multivariate analysis of the effect of X-Ray rating and smoking habits on lung cancer risk. Smoking habits were modelled as before. Both an additive and multiplicative model were used in the analysis, but only the results of using an additive model are presented here.

Also using this analytical technique, smoking was the most powerful risk factor. Being a member of sub-cohort B or C was also associated with an increased lung cancer risk (P=0.01) when smoking was not taken into account. The association of X-Ray rating with increased lung cancer risk could, however, no longer be demonstrated if a smoking variable was included in the model.

Effect of Aluminum Prevention :

The possible effect of exposure to aluminum prevention was investigated both in the cohort study and in the case-control

study. Most men in the gold miners' cohort under study participated at some time during their gold mining experience in aluminum prevention of silicosis, which was introduced into Ontario gold mines in the mid-1940s. Of the total of 7057 members of the gold miners' cohort, 6730 participated at some time in aluminum prevention and 327 never did.

Table 18 compares the relative lung cancer risk of men ever exposed to aluminum prevention with those never exposed to aluminum dust while gold miners.

The lung cancer risk is significantly elevated in both the group exposed to aluminum and the group not so exposed. The difference in risk between the two groups is statistically not significant.

Table 19 compares the distribution of lung cancer cases by cell type in nickel/copper miners and in gold miners using the reclassification of the cancers by cell type.

DISCUSSION

The conditions for inclusion in the gold miners' cohort have remained largely unchanged since the 1983 report on the Ontario Miners Mortality Study. One change, however, has been made. As indicated earlier, the new requirement was that the man had worked as a miner some time between 1955 and 1977. The earlier requirement was that the man was known to have been alive at some time during this time period, as indicated by attending a miners' examination during 1955 to 1977. This change was made in order to eliminate any possible bias resulting from the self-selection of men who attended miners' examinations at a time after leaving active mining when these examinations were no longer required.

The change in conditions for inclusion in the study cohort resulted in both fewer observed and expected deaths from the various causes as compared to the earlier report. This change did not affect relative risk in any substantial way with the notable exception of two causes of death: the number of deaths from silico-tuberculosis declined from 41 in the earlier report to 23 in this report. Also the number of deaths assigned to silicosis and chronic interstitial pneumonia declined, as a result of the changed conditions for inclusion of gold miners in the study, from 111 deaths reported earlier to 39 deaths. Men suffering from silicosis with or without complicating tuberculosis, were obviously

more likely to attend miners' examinations after stopping work in mines, than men with no such disability. The change in the definition of a gold miner for the purpose of the study did not affect the estimated relative risk for most other causes, including cancer of the trachea, bronchus and lung, cancer of the stomach, violent deaths and other causes.

Including, as was done in the 1983 report, a group of self-selected men in the study who did not actually work in mines at any time between January 1, 1955 and December 31, 1977, did in fact introduce a bias in relation to silicosis, whether complicated by tuberculosis or not. However, it did not cause an appreciable bias in relation to other causes of death.

The primary aim of the study was to investigate to what extent the observed increased lung cancer risk in gold miners was job-related or the result of risks that were unrelated to the mining occupation of the men. For this reason, the possible effect of both, factors not directly related to the occupation of the men and factors directly related to the mining occupation, were investigated.

The Effect of Smoking :

Among the non-occupational environmental factors most closely associated with lung cancer risk is cigarette smoke, which is a well-recognized cancer-causing agent. The effect of

cigarette smoke on lung cancer risk was also documented in the gold miners' cohort. Table 11, derived from the case-control study, indicates a relative lung cancer risk of smokers as compared to non-smokers of a factor of 7. Cigarette smoke is by far the most important agent causing lung cancer in the gold miners' cohort. This in itself, however, does not mean that the higher risk of lung cancer in gold miners compared with nickel/copper miners is the result of differences in smoking habits of the two miners' cohorts. The results of a comparison of smoking habits of gold miners and nickel/copper miners given in Table 3 do not indicate that gold miners smoke more than nickel/copper miners. Differences in lung cancer rates between the two cohorts are therefore unlikely to be the result of differing smoking habits. When further investigating any possible association between a potential cancer causing agent in the working environment and lung cancer risk, smoking habits of the individual miners have to be taken into consideration.

Attained Age and Lung Cancer Risk in Gold Miners :

A possible explanation of the higher lung cancer risk in gold miners as compared to nickel/copper miners could be differences in age distribution of the two populations. Table 10, however, indicates that a higher than expected lung cancer risk in gold miners is not limited to one specific age group. All ages normally affected by lung cancer show a significantly increased risk. In fact, the middle age

group, 55 to 64 years, that is well represented in both cohorts, shows the highest relative risk.

There are no major differences in the socio-economic status of the two miners' cohorts and no major differences in other life-style factors are to be expected. As a result it was considered unlikely that the difference in lung cancer risk between nickel/copper miners and gold miners can be explained by major differences in life style. It was, therefore, considered appropriate to investigate associations of exposure to possible cancer causing agents in the mine environment and lung cancer risk.

Possible Cancer Causing Agents in the Mine Environment :

Before attempting to investigate which specific environmental factors in the mines might be associated with increased lung cancer risk, the more basic question of a possible association of exposure to the mine environment in general and lung cancer risk was investigated.

Years of Exposure and Lung Cancer Risk :

The relationship between years of exposure and lung cancer risk is the simplest, most commonly used and often misleading measure of an association between exposure to a possibly harmful work environment and risk. "What is so frequently overlooked is the singular fact that the use of duration of exposure is a meaningful surrogate of true dose

only if all exposed subjects concerned in the dose-response analysis were subjected to uniform exposure for different periods." (Jo86) As Table 4 indicates, there is no clear relationship between the total number of years of underground work and relative lung cancer risk. This in itself, however, does not indicate that the higher relative lung cancer risk in gold miners is not work-related. Total years of work underground might not be an appropriate indicator of exposure, because of the great changes of exposure rate over the several decades that members of the gold miners' cohort worked in the mines. For this reason, the period of starting gold mining or the number of years of underground work prior to the mid-1940s, when major improvements of the mine environment were made, might provide a more meaningful measure of association with lung cancer risk than the total number of years worked underground.

Period of Starting Gold Mining :

Working conditions in Ontario gold mines have greatly improved over the last several decades during which members of the gold miners' cohort have been employed in the mines. Over this period, dust suppression techniques and ventilation have lowered dust concentrations by a factor of the order of 10. The organization of blasting operations has changed over the years, greatly diminishing the exposure of miners not only to blasting fumes, but also to dust. It is, therefore, not unlikely that if increased relative lung

cancer rates in gold miners are work related, the calendar period of underground employment might provide a more meaningful indicator of exposure than years of employment. Work in the early years generally entails greater exposure than work in the later years.

The data in Table 5 indicate that both men under and over the age of 65 years, who started underground gold mining prior to the year 1936, show a significantly increased lung cancer risk. In addition, in men who started underground gold mining between 1936 and 1944, the relative lung cancer risk in the age group below 65 years is borderline significantly larger than 1 ($P = 0.07$). In sub-groups starting gold mining later, the difference between the number of expected and observed lung cancer deaths was no longer significant.

In men who started gold mining early, there is, of course, also a longer time interval between the time of starting mining gold and the time of observation. Both increased risk with greater exposure, or with increasing latent interval from start of exposure to the end of follow-up, clearly point to a work-related risk.

Years of Dust Exposure in Mines Prior to 1945 :

As indicated earlier, the total number of years worked in dust exposure underground is not necessarily closely related to the total exposure of a man. One year's work in the early years might well lead to the same uptake of contaminants from

the mine atmosphere as 10 years of work in the later period. Major improvements in the mining environment were achieved in the mid 1940's.

The lung cancer risk declined significantly with more recent entry into the gold mines and increased with increasing years of dust exposure prior to 1945 (see Tables 5 and 6). Dust exposure in the later years contributed only very little to lung cancer risk because of the greatly diminished exposure to the cancer causing agent or possibly because the time interval between exposure and the period of observation was too short.

The failure to demonstrate any major differences in life style, including differences in smoking habits, between the gold miner and nickel/copper miner cohorts, makes an association of life style factors with increased lung cancer risk in gold miners unlikely. Both the increased lung cancer risk in "early starters" as compared to "late starters", and the increasing risk of lung cancer with an increasing number of years of dust exposure in the mines prior to 1945, clearly point to an occupational risk.

The primary objective of the study was to determine if the increased lung cancer risk in gold miners is related to life style or if conditions in the working environment significantly contributed to the observed risk. No difference in life style factors between gold miners and nickel/copper miners could be demonstrated. There is, however, a

significant association of both year of first gold mining and years of dust exposure prior to 1945 with lung cancer risk after taking into account the effect of age and calendar period. These findings support the hypothesis that the increased lung cancer risk observed in gold miners is work related.

The secondary objective of the study was to investigate associations of the observed lung cancer risk with specific pollutants or health conditions related to these pollutants in the mine environment.

Silicosis and Lung Cancer :

Several experimental and epidemiological studies carried out in recent years point to an association between silicosis and lung cancer risk.

A number of hypotheses are examined to explain this fact:

1)Silica itself is a lung carcinogen and silicosis resulting from the exposure increases lung cancer risk.

2)Silica enhances the effect of carcinogens inhaled simultaneously.

3)Both the risk of silicosis and lung cancer are enhanced by smoking.

4)Silicosis is an indicator of heavy exposure to dust containing silica as well as possibly other substances that might be carcinogenic.

Dagle (Da86) observed epidermoid carcinoma in the lungs of rats having inhaled silica dust. The tumors appeared in 10 out of 53 female rats surviving longer than 494 days and in

1 out of 47 male rats, indicating a sex difference in sensitivity to silica exposure, taking lung cancer as the endpoint.

The first hypothesis thus finds support from experimental data on rats. The effect, however, appears to be sex-dependent.

In addition there are also marked species differences in sensitivity to silica. Saffiotti (Sa86) reviewed the new epidemiological evidence of carcinogenic activity of crystalline silica and notes the marked species difference in this regard. While inhaled or instilled silica causes both fibrosis and lung cancer in rats, instilled silica particles remain phagocytosed in macrophages and produce neither necrosis of these phagocytes nor subsequent fibrosis or tumors in Syrian golden hamsters.

A number of epidemiological studies demonstrated an increased lung cancer risk in silicotics. Finkelstein and co-workers (Fi82,Fi86) compared the lung cancer risk of silicotics, mostly gold miners, to the lung cancer risk in the male population of Ontario and found an increased risk in the silicotic group.

Westerholm and co-workers (We86) found an increased lung cancer risk in silicotics employed in mining, quarrying and tunneling and in silicotics employed in iron and steel foundries. The increased lung cancer risk in silicotics still persisted in the first occupational group when exposed

non-silicotics were used as controls. Silicotics from iron and steel foundries, however, showed no significant increase in lung cancer rates over non-silicotics similarly exposed.

Neither of the two studies mentioned above controlled for smoking habits.

No significant increase in lung cancer risk was detected by Brown and co-workers (Br86) in underground gold mine workers. The authors point out, however, that U.S. mortality rates were used to generate the expected number of lung cancer deaths, rather than South Dakota rates, where the majority of the cohort resided. If South Dakota rates had been used rather than U.S. rates, the SMR for cancer of the trachea, bronchus and lung could have been elevated by as much as 38 percent. Smoking data on individual miners were not considered, but it should be noted that smoking was prohibited in the mine from the early 1900s to 1952 and this rule was, according to the authors, strictly enforced because of a fire hazard. It is, therefore, quite possible that these miners smoked, on the average, less than the control population.

The Experience in Ontario Gold Miners :

Ontario gold miners have been shown to have a significantly higher lung cancer risk than an appropriately matched male population of Ontario and also greater than a matched cohort of nickel/copper miners. This increased risk can be

demonstrated in all three sub-cohorts by X-Ray rating (see Table 7). The data in Table 7 also indicate that the lung cancer risk is not independent of sub-cohort, but increases from lowest (sub-cohort A) to highest X-Ray rating (sub-cohort C).

Also the data derived from the case-control study indicate that members of sub-cohorts B and C have a significantly greater risk of dying of lung cancer than members of sub-cohort A. The relative risk is 2.1 and the P-value is 0.02 (see Table 13). Also a multivariate analysis (see Table 17) indicates a significant increase of lung cancer risk with increasing X-Ray rating if no term for smoking is included in the model.

All these data indicate an increased risk in sub-cohorts B and C as compared to sub-cohort A. In this respect the data derived from this study support a hypothesis that silicosis increases lung cancer risk.

Guidotti and Cohen (Gu86) expand the earlier hypothesis proposed by Cohen in 1978, that smoking might produce "pulmonary dysfunction", a lesion that might affect clearance from the lung of substances that might initiate carcinogenesis. They propose that silica might act in a similar way as smoking, affecting not only mucociliary clearance, but also exerting macrophage toxicity as well as other effects that might enhance both retention and activation of potential carcinogens.

It is possible that smoking could lead to a pulmonary insult resulting in pulmonary dysfunction, causing airflow obstruction, increased retention of both silica and possible cancer causing agents in the lung, thus increasing both silicosis and lung cancer risk.

Hessel and co-workers (He86) carried out a case-control study of men, members of a "Gold Miners Provident Fund". The cases were members of the Fund who died of lung cancer between 1974 and 1983. The controls were members of the same fund, matched by year of birth +/- 2 years and matched for smoking habits. Silicotics showed in this study no greater lung cancer risk than non-silicotics.

When comparing smoking habits of gold miners included in this study by sub-cohort, there is an indication that members of sub-cohort C smoke more than members of sub-cohorts A and B. This finding (see Table 20) tends to support a hypothesis that smoking increases not only lung cancer risk, but also the risk of acquiring silicosis.

Table 14, derived from the nested case-control study indicates a relative risk of 1.3 (P= 0.5) of being a member of sub-cohort B or C if both case and control are smokers. It should be borne in mind, however, that the sample size was greatly reduced in Table 14 and thus some discriminating power was lost. The multivariate analysis summarized in Table 17 indicates that the observed strong association between X-Ray rating, as represented by membership in one of

the three sub-cohorts of miners, and lung cancer risk diminishes or disappears if a variable representing smoking habits is included in the model.

The hypothesis that smoking increases both lung cancer risk and the risk of silicosis is supported by the data presented in this study and it is biologically plausible. The observed greater lung cancer risk in silicotics as compared to non-silicotics observed in this study, as well as in other studies, is likely to be totally or at least partially due to the differences in smoking habits between the sub-cohorts or between silicotics and non-silicotics. Smoking alone does not produce presilicotic lesions characterized by the Ontario X-Ray code 4 or the ILO code 0/1 or 1/0. This type of X-Ray finding is rare in nickel/copper miners whose smoking habits are similar to gold miners' but who are exposed only to low levels of free silica.

Silica could possibly enhance the effect of other carcinogens inhaled simultaneously.

Niemeier and co-workers (Ni86) failed, as did Holland, to produce respiratory tract tumors by instillation of silica particles into the trachea of Syrian golden hamsters. However, silica dust in combination with BaP produced more lung cancers in Syrian golden hamsters than BaP in saline alone. Silica enhanced the effect of another carcinogen.

This might not necessarily be a specific effect of silica

but a number of other particulates might have a similar effect, perhaps facilitating the transport of carcinogens into macrophages.

Silicosis can also be viewed as an indicator of heavy exposure to dust in mines where silica is a constituent of the ore dust.

The Relation of Individual Environmental Factors and Their Interaction on Lung Cancer Risk :

Individual environmental factors examined include :

Cigarette smoke;
Years in dust exposure;
Years in high dust exposure;
Years in high arsenic exposure when dust levels were high;
Years in ore with elevated amounts of fibrous materials when dust levels were high;
Years in high sulfide levels when dust levels were high;
Years in high silica levels when dust levels were high;

Table 16 gives some of the results of the analysis.

The analysis of the data suggests an association of lung cancer risk with high levels of arsenic and fibers in the ore when dust levels were high. No suggestion of such an association was found for high silica in the ore when dust levels were high. The appropriate values of Chi-square and P-values are given in Table 16. It should be noted that exposure data were of low quality, and could be used only in qualitative terms. This greatly diminishes the discriminatory power of the analysis. In gold miners who never worked in a gold mine with high arsenic or fiber levels in the ore, the lung cancer risk is still significantly higher than expected. It may well be possible that other factors, such

as the short lived progeny of radon-222, might be responsible for the increase in risk.

Data on mines, where higher levels of radioactivity in the ore could be expected, were obtained from the Geological Survey of Ontario. No association was found between months worked in these mines and elevated lung cancer risk. It should, however, be noted that there is not necessarily a close association between low levels of radioactivity in the ore and concentrations of radon progeny in the mine atmosphere. Radon and radon daughter levels might well be elevated in gold mines, where the geology does not predict it.

Several facts have to be considered, however, when evaluating these results.

The quality and number of dust measurements in the mines, particularly in the early years, were not sufficient to attach exact numbers to the dust counts. It was decided to use, therefore, only qualitative statements as "high" or "low". The exposure to dust was also dependent on the job the man actually carried out underground. It can be assumed that misclassification did occur in the exposure data. The result of such misclassification is likely to be a certain degree of loss of discrimination and less pronounced associations are likely to be missed.

When subdividing the data into increasingly finer sub-groups, the number of available data points decreases and it becomes

more difficult to obtain significant results. The association of months of exposure to high levels of dust is, as a result, based on far more data than the combination of high dust and high sulfide or arsenic levels. In addition, high arsenic levels in the ore are, as a rule, associated with high sulfide levels. The two constituents in the ore, therefore, are not entirely independent. Based on a priori knowledge, it is not unlikely that high dust in combination with high arsenic levels in the ore, is considered suspect, rather than high dust in combination with high sulfide levels in the ore. Fibrous materials such as asbestos fibres are, in combination with smoking, known carcinogens.

It is noteworthy, that the analysis of the data did not suggest an association of increased risk of lung cancer with exposure to high dust levels combined with high levels of silica in the ore. This might possibly be explained by the fact that the levels of free silica in the ore of the three mining camps are not sufficiently different or perhaps by the assumption that exposure to silica is not associated with increased lung cancer risk. The data on Ontario gold miners do not, however, indicate an association of exposure to silica with increased lung cancer risk.

Effect of Aluminum Prevention :

The data in this study do not indicate any increase in lung cancer risk resulting from exposure to aluminum dust used for the prevention of silicosis. The number of gold miners

not exposed to aluminum prevention is relatively small and the results have therefore to be taken with a degree of caution. It is, however, noteworthy that neither the cohort study nor the case-control study provide any indication of aluminum prevention increasing the lung cancer risk. Men who never were exposed to aluminum prevention have a significantly increased lung cancer risk as compared to non-miner controls (see Table 18) and the increased lung cancer risk in gold miners cannot be explained by exposure to aluminum dust.

Men who started gold mining in Ontario before 1936, or some ten years prior to the introduction of aluminum prevention into Ontario gold mines, show a higher relative lung cancer risk than those who started gold mining after 1935. This supports the hypothesis that aluminum prevention is not responsible for the increased lung cancer risk observed in Ontario gold miners.

In an earlier report (Mu83), an increased death rate of stomach cancer was found in gold miners as well as a diminished rate of cancer of the colon and rectum. These findings could be possibly explained by a high proportion of immigrants in the gold miners' cohort.

Newman and Spengler (Ne84) examined the cancer rates in the immigrant population of Ontario and compared them to the rates in Ontario residents born in Ontario. They found that Ontario residents born in the United Kingdom, Italy, the

Netherlands, Poland and in the USSR have a significantly higher risk of dying of stomach cancer than Ontario born residents of the province. On the other hand, Ontario residents born in Italy and the USSR have a significantly lower risk of dying of cancer of the colon and rectum than Ontario born residents.

Among the 54 Ontario gold miners who died from 1955 through 1977 of cancer of the stomach, there were 24 (44 percent) born in North America. Of those remaining, 24 were known to have been born in other countries. A total of 15.60 stomach cancer cases were expected among gold miners born in Canada, giving an SMR of 154. The expected number of stomach cancer deaths among gold miners born outside Canada was 17.54, giving an SMR of 137. The increased death rate from stomach cancer in the gold miners cohort therefore cannot be explained by the high proportion of immigrants in this cohort. No increased risk of stomach cancer was observed among nickel/copper miners.

No explanation for these findings is being offered at present.

A comparison of the classification of lung cancers by cell type carried out in hospitals and their reclassification by Prof. Ritchie showed major discrepancies. These will be discussed in a separate publication. For the purpose of this study, only the results of the reclassification of lung cancers by cell type were considered. There were no major

differences in the distribution of lung cancers by cell type between gold miners and nickel/copper miners (see Table 19).

The purpose of the study was to determine if the observed increased lung cancer rates in gold miners compared to the male population of Ontario and to nickel/copper miners was related to life-style differences or to factors in the mine environment. Should it be found that the increased risk was the result of exposure in the mine environment, an attempt was to be made to identify the causative agent or agents.

A change in the definition of underground gold miners, made necessary because of a bias introduced in the earlier definition used in the 1983 report, greatly reduced the number of deaths from silico-tuberculosis and silicosis in the underground gold miners' cohort. The relative risk of dying of other causes, including cancer of the lung, remained virtually unchanged.

CONCLUSIONS

1) It is most unlikely, that the increased lung cancer risk in Ontario gold miners was caused by differences in life style, including differences in smoking habits, between gold miners, nickel/copper miners and an appropriately matched male population of Ontario.

2) The data indicate that factors in the underground mine environment are responsible for the increase in lung cancer rates in gold miners.

3) Both early start of underground work in Ontario gold mines and years of dust exposure in these mines prior to 1945 are associated with increased lung cancer risk. Later exposure contributes only very little to risk.

4) The data indicate a positive, significant association between X-Ray rating and lung cancer risk. This association largely disappears after correction for differences in smoking habits of silicotic compared to non silicotic gold miners. In an environment producing silicosis, smoking increases both the silicosis and lung cancer risks.

5) Silicosis in Ontario gold miners is not only related to smoking habits, but also is an indicator of high exposure to dust containing free silica and any other cancer causing agents that might be present in it.

6) A weak association of lung cancer risk with exposure to high levels of dust combined with exposure to high levels of

arsenic in the dust was found.

7) No association of lung cancer risk in gold miners with exposure to the short-lived progeny of radon-222 could be investigated, as no historic data on radon daughter levels in the gold mines are available. No association of lung cancer risk with radioactivity in the mines predicted from the geology could be detected.

8) No association of lung cancer risk in gold miners with exposure to aluminum prevention could be detected.

9) There were no major differences in the distribution of lung cancers by cell type between gold miners and nickel/copper miners.

10) No causative agent for the increased stomach cancer rate in gold miners was identified.

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TABLE 1

MORTALITY DATA
UNDERGROUND GOLD MINERS

Cause of Death	Observed[1]		Expected	SMR	P-value
All Causes	1695		1787.4	95	
All Disease Causes	1477		1663.9	89	
Infective Diseases	23	(14.6-34.5)	12.3	187	4×10^{-3}
Silicotuberculosis	14				
Respiratory Tuberculosis	4		4.6	87	
Other Tuberculosis	0		1.1	0	
All Other Infective Diseases	5		6.1	82	
Malignant Neoplasms	372		384.1	97	
Digestive Organs	117		132.6	88	
Stomach	54	(40.6-70.5)	34.3	157	1×10^{-3}
Intestine, Rectum	27		55.7	48	
Other Digestive	36		42.5	85	
Respiratory System	171	(146.3-198.7)	124.9	137	2×10^{-5}
Nose, Nasal Cavities etc	1		1.0	102	
Larynx	5		5.5	92	
Trachea, Bronchus, Lung	165	(140.8-192.2)	117.5	140	6×10^{-6}
Other	0		1.0	0	
Lymphatic and Haematopoietic Tissue	24		33.5	72	
Other	60		93.2	64	
Bone	0		1.6	0	
Kidney	8		9.1	88	
Bladder	6		14.0	43	
Brain	9		11.0	82	
Prostate	17		26.4	64	
Skin	1		4.3	23	
All Other	19		26.9	71	
Diseases of Circulatory System	782		984.0	79	
Ischaemic Heart Disease	549		723.1	76	
Cerebrovascular Disease	126		137.7	92	
Other	107		123.3	87	
Diseases of Respiratory System	159	(135.3-185.7)	113.6	140	1×10^{-5}
Influenza, Pneumonia, Bronchitis, Asthma	104		94.1	111	
Silicosis, Chronic Interstitial Pneumonia	39	(27.7-53.3)	5.5	705	$< 10^{-12}$
Other	16		14.0	114	
All Other Disease Causes	141		169.9	83	
Accidents, Poisoning, Violence	218	(190.0-248.9)	123.4	177	$< 10^{-12}$
Motor Vehicle Traffic Accidents	32		36.3	88	
Water Transport Accidents	12	(6.2-21.0)	3.9	309	7×10^{-4}
Accidental Falls	22	(13.8-33.3)	14.8	149	5×10^{-2}
Motor Vehicle Non-Traffic Accidents	0		1.0	0	
Accidental Drowning and Submersion	4		4.2	95	
Inhalation or Ingestion of Food Causing Obstruction	2		2.5	79	
Blow from Falling or Projected Missile	49	(36.2-64.8)	2.7	1797	$< 10^{-12}$
Firearms or Explosives	11	(5.5-19.7)	1.3	872	1×10^{-7}
Other Vehicle Accidents	4	(1.1-10.2)	0.8	529	7×10^{-3}
All Other Accidents	82	(65.2-101.8)	56.1	146	7×10^{-4}

Note: [1] 95% confidence limits on the observed number of deaths are shown in brackets.

TABLE 2

RELATIVE RISK OF GOLD MINERS VERSUS NICKEL/COPPER MINERS

Cause of Death	Ages less than 70		Ages Over 70	
	Relative Risk (%)	P-Value	Relative Risk (%)	P-Value
Digestive Cancers	115	N.S.	82	N.S.
Stomach Cancers	238	1×10^{-3}	78	N.S.
Lung Cancers	190	3×10^{-5}	67	N.S.
All Other Cancers	82	N.S.	78	N.S.
Diseases of Circulatory System	90	N.S.	91	N.S.
Diseases of Respiratory System	186	6×10^{-4}	152	N.S.
All Other Diseases	109	N.S.	138	N.S.
All Violent Deaths	140	6×10^{-4}	153	N.S.

Note: Strata are age groups.
Summary Relative Risk was calculated using Mantel-Haenszel weights.

N.S. means not significantly different from 1 (the P-value was greater than 0.05).

TABLE 3

SMOKING HABITS OF GOLD AND NICKEL MINERS

Year of Birth Group	GOLD						NICKEL					
	Never Smoker	Ever Smoker				No Smoking Information	Never Smoker	Ever Smoker				No Smoking Information
		Amount Smoked (Packs/Day)						Amount Smoked (Packs/Day)				
		Unknown	< ½	½-1½	>1½			Unknown	< ½	½-1½	>1½	
<1910	23(25)	15(16)	19(21)	33(36)	2(2)	55	8(26)	9(29)	2(6)	11(35)	1(3)	33
1910 - 1919	13(19)	8(12)	11(16)	34(49)	3(4)	24	17(19)	14(16)	12(13)	44(49)	2(2)	20
1920 - 1929	8(15)	4(8)	10(19)	24(45)	7(13)	22	13(10)	21(16)	22(17)	68(52)	8(6)	19
1930+	1(5)	1(5)	4(19)	13(62)	2(9)	11	8(10)	11(14)	7(9)	42(54)	10(13)	14
Total	45(19)	28(12)	44(19)	104(44)	14(6)	112	46(14)	55(17)	43(13)	165(50)	21(6)	86

Note: Percentages are in parentheses

TABLE 4

LUNG CANCER RISK BY NUMBER OF YEARS OF DUST EXPOSURE IN GOLD MINES

Years Worked	Observed	Expected	SMR	P-value
≤10	23	16.0	144	6×10^{-2}
11 - 20	43	31.6	136	3×10^{-2}
21 - 30	54	40.1	135	2×10^{-2}
31 +	45	29.9	150	6×10^{-3}
TOTAL	165	117.6	140	2×10^{-5}

TABLE 5

LUNG CANCER RISK IN UNDERGROUND GOLD MINERS (BY CALENDAR PERIOD OF START OF GOLD MINING AND AGE GROUP)

Age up to 65 years

Year Started*	Observed	Expected	SMR	P-Value
1	42	21.6	195	6×10^{-5}
2	25	18.0	139	7×10^{-2}
3	26	20.0	130	1×10^{-1}
	93	59.6	156	4×10^{-5}

Age 65 + years

Year Started*	Observed	Expected	SMR	P-Value
1	56	40.4	139	1×10^{-2}
2	14	13.5	103	N.S.
3	2	4.1	49	N.S.
	72	58.0	124	4×10^{-2}

Note * 1 - started before 1936

2 - started between 1936 and 1944

3 - started after 1944.

TABLE 6

LUNG CANCER RISK BY NUMBER OF YEARS OF DUST EXPOSURE IN GOLD MINES PRIOR TO 1945

Years in Dust Exposure	Observed	Expected	PYR	SMR
0	28	24.1	54901	116
0.1 - 5	26	18.7	16905	139
5.1 - 10	38	26.7	16694	143
10.1 - 15	35	26.2	13048	134
15.1 - 20	27	16.6	7424	163
20.1+	11	5.4	2296	202
TOTAL	165	117.6	111267	140

TABLE 7

LUNG CANCER RISK BY X-RAY RATING

Sub-cohort	Observed	Expected	SMR	P-Value
A	113	89.8	126	1×10^{-2}
B	34	21.1	161	6×10^{-3}
C	18	6.7	267	2×10^{-4}

POISSON REGRESSION ANALYSIS OF LUNG CANCER RISKS BY SUBCOHORT LOG - LINEAR RISK FUNCTION

Model for Log-linear Relative Risk	Deviance	df
a	8.08	2
a + b x sub-cohort	0.37	1

NOTE: Sub-cohort A is coded as -1, Sub-cohort B as 0 and Sub-cohort C as 1.
The hypothesis that all sub-cohorts have the same relative risk is not accepted.
(x^2 = 8.08, 2 df, P = 0.02) and the hypothesis of no increase in risk with sub-cohort is not accepted (x^2= 8.08 - 0.37 = 7.71, 1 df, P = 5×10^{-3}).

TABLE 8

COMPARISON OF SUB GROUPS OF MINERS BY YEAR OF START AND BY X-RAY RATING

AGE GROUP UNDER 65 YEARS

Year Start		Sub-Cohort A	B	C	Total	P-values Comparing Sub-cohorts (A B)	(A C)	(B C)
before 1936	Observed	27	10	5	42			
	Expected	15.3	5.0	1.2	21.6	4×10^{-1}	9×10^{-2}	2×10^{-1}
1	PYR	13885	3962	898	18745			
	SMR	176	200	402	195			
	P-Value	4×10^{-3}	3×10^{-2}	9×10^{-3}	6×10^{-5}			
1936 - 1944	Observed	22	3	0	25			
	Expected	16.2	1.7	0.2	18.0			
2	PYR	19843	1521	151	21515	4×10^{-1}		
	SMR	136	180	0	139			
	P-Value	1×10^{-1}	2×10^{-1}		7×10^{-2}			
1945 or later	Observed	26	0	0	26			
	Expected	19.2	0.80	0.1	20.0			
3	PYR	52531	1104	61	53696			
	SMR	136	0	0	130			
	P-Value	8×10^{-2}			1×10^{-1}			
Total	Observed	75	13	5	93			
	Expected	50.7	7.5	1.5	59.6			
	PYR	86259	6587	1110	93956	3×10^{-1}	8×10^{-2}	2×10^{-1}
	SMR	148	174	333	156			
	P-Value	8×10^{-4}	4×10^{-2}	2×10^{-2}	4×10^{-5}			
P-Values Comparing Year Of Start Group	1 and 2	2×10^{-1}	6×10^{-1}		1×10^{-1}			
	1 and 3	2×10^{-1}			7×10^{-2}			
	2 and 3	6×10^{-1}			5×10^{-1}			
	1 vs. 2 and 3 inclusive	2×10^{-1}			5×10^{-2}			

Note: One sided P-values are shown

TABLE 9

COMPARISON OF SUB GROUPS OF MINERS BY YEAR OF START AND BY X-RAY RATING

AGE GROUP 65 YEARS AND OVER

Year Start		Sub-Cohort A	B	C	Total	P-values Comparing Sub-cohorts (A B)	(A C)	(B C)
before 1936	Observed	26	19	11	56			
	Expected	24.4	11.3	4.7	40.4	9×10^{-2}	3×10^{-2}	3×10^{-1}
1	PYR	7384	3396	1405	12185			
	SMR	107	169	233	139			
	P-Value	4×10^{-1}	2×10^{-2}	9×10^{-3}	1×10^{-2}			
1936 - 1944	Observed	10	2	2	14			
	Expected	11.1	2.1	0.4	13.5	6×10^{-1}	6×10^{-2}	1×10^{-1}
2	PYR	3225	584	112	3921			
	SMR	90	98	513	103			
	P-Value	4×10^{-1}	7×10^{-1}	6×10^{-2}	5×10^{-1}			
1945 or later	Observed	2	0	0	2			
	Expected	3.6	0.3	0.1	4.1			
	PYR	1065	99	39	1203			
3	SMR	55	0	0	49			
	P-Value	3×10^{-1}			2×10^{-1}			
Total	Observed	38	21	13	72			
	Expected	39.1	13.6	5.2	58.0	6×10^{-2}	5×10^{-3}	1×10^{-1}
	PYR	11674	4079	1556	17309			
	SMR	97	154	248	124			
	P-Value	5×10^{-1}	4×10^{-2}	3×10^{-3}	4×10^{-2}			
P-Values Comparing Year Of Start Group	1 and 2	4×10^{-1}	4×10^{-1}	3×10^{-1}	2×10^{-1}			
	1 and 3	3×10^{-1}			9×10^{-2}			
	1 and 3	4×10^{-1}						
	1 vs 2 and 3 inclusive	3×10^{-1}			8×10^{-2}			

Note: One sided P-Values are shown.

TABLE 10

OBSERVED AND EXPECTED DEATHS BY AGE GROUPS

Age group (Years)	Observed	Expected	SMR	p-value
45-54	21	14.3	147	6×10^{-2}
55-64	70	43.3	161	1×10^{-4}
65 +	72	58.0	124	4×10^{-2}

TABLE 11

GOLD CASE CONTROL STUDY

Effect of smoking on lung cancer risk

		CASES	
		Non-Smoker	Smoker
CONTROLS	Non-Smoker	5	42
	Smoker	6	66

RELATIVE RISK OF A SMOKING IS 42/6 = 7

Chi square = 27 $P \ll 0.001$

Smoker means smoked within 10 years of death and non-smoker means did not smoke within 10 years of death.

TABLE 12

CATEGORIES OF EMPLOYMENT YEARS IN HIGH DUST LEVELS ASSOCIATED WITH HIGH LEVELS OF INDIVIDUAL CONTAMINANTS IN THE ORE

Type of Contaminant	Cases			Controls		
	No of men	Mean	Sum	No of men	Mean	Sum
Silica	4	9.7	38.6	7	2.0	13.7
Fibers	7	5.2	36.2	8	1.7	13.6
Arsenic	10	5.2	52.3	6	1.4	8.2
Sulphides	32	6.4	204.8	30	4.0	120.7

TABLE 13

COMPARISON OF X-RAY RATING CASES AND CONTROLS

		CASE	
		A	B + C
CONTROL	A	64	30
	B + C	14	11

Rel. Risk = 2.1
Chi square = 5.8 P = 0.02

TABLE 14

SMOKING AND X-RAY CATEGORY IN CASES AND CONTROLS

Both Case and Control Are Current Smokers

		Cases	
		A	B or C
Controls	A	34	14
	B or C	11	7

RR = 1.3

Note: Current Smoker means smoked in the 10 years before date of death or notional date of death.

$X^2 = 0.36$ P = 0.5

TABLE 15

MONTHS OF EXPOSURE TO BLASTING FUMES

	CASES			CONTROLS		
Category	Number	Average	Total	Number	Average	Total
I	41	43.6	1,789	35	41.7	1,458
II	64	60.3	3,859	69	61.6	4,253
III	85	53.4	4,538	85	54.0	4,588
IV	99	113.2	11,206	94	125.3	11,774

TABLE 16

SUMMARY OF RESULTS OF CASE-CONTROL STUDY

MODEL RR= 1 + a x Current Smoker + b x Months in Exposure

Exposure	Parameter Estimate a	Parameter Estimate bx100	Chi Square for extra term	P-Value
Smoking	6.0		30.37	$< 10^{-6}$
Months in underground gold	13.2	0.6	0.46	0.5
High Dust	9.0	3.1	4.17	0.04
High Dust High Arsenic	6.3	9.2	2.89	0.09
High Dust High Sulphides	9.0	3.4	4.40	0.04
High Dust High Fibres	7.0	1.6	2.26	0.1
High Dust High Silica	6.0	0.1	0.01	0.9

Notes: Current smoker means smoked within 10 years of death or notional date of death. Current smoker was coded as 1 and not current smoker was coded as 0.
RR = Relative Risk

TABLE 17

ANALYSIS OF EFFECTS OF X-RAY CATEGORY AND SMOKING ON LUNG CANCER RISK

Model RR = 1 + a Current Smoker + b X-Ray Category + c Smoking x X-Ray Category.

Parameter Estimates				Model Chi Square	df	Chi Square for Extra Term	P-Value
a	b	c					
0	1.1	0	-	5.96	1		0.01
6.0	0	0		30.37	1		$< 10^{-6}$
6.0	0.9	0		30.83	2	0.46	0.5
5.3	0.3	2.7		31.58	3	0.75	0.4

Notes: Current smoker means smoked within 10 years of death or notional date of death. Current smoker was coded as 1 and not current smoker was coded as 0.

RR = Relative Risk

X-Ray category was 0 if the man never became X-Ray subcohort B or C up to death of case or notional date of death of control. Otherwise X-Ray category was 1.

TABLE 18

EFFECT OF ALUMINUM PREVENTION OF SILICOSIS

	Observed	Expected	SMR	P-Value
Ever Aluminum	151	109.75	138	$1x10^{-4}$
Never Aluminum	14	7.87	178	$3x10^{-2}$

TABLE 19

DISTRIBUTION OF LUNG CANCER CASES BY CELL TYPE IN NICKEL/COPPER MINERS AND IN GOLD MINERS.

	Nickel/copper		Gold	
Cell type of cancer	Number	Percent	Number	Percent
Adeno carcinoma	9	24	19	23
Squamous cell	8	22	23	28
Small cell	11	30	25	31
Large cell	8	22	13	16
Other	1	3	1	1
Total re-evaluated*	37	101	81	99

*The Total number of lung cancer deaths was:

Nickel/copper miners: 83 re-evaluated 45%
Gold miners: 165 re-evaluated 49%

TABLE 20

COMPARISON OF SMOKING HABITS BY X-RAY RATING

X-Ray Rating : Subcohorts

Year of Birth Cohort	A				B				C			
	Never Smoker		Ever Smoker		Never Smoker		Ever Smoker		Never Smoker		Ever Smoker	
	No	%	No	%	No	%	No	%	No	%	No	%
1885 - 1899	5	29	12	71	2	40	3	60	1	10	9	90
1900 - 1904	8	22	19	78	4	36	7	64	1	7	14	93
1905 - 1909	6	21	23	79	3	50	3	50	1	8	12	92
1910 - 1914	8	28	21	72	4	36	7	64	1	10	9	90
Subtotal	27	26	75	74	13	39	20	61	4	8	44	92
1915 - 1937	14	14	85	86	0	0	15	100	0	0	8	100
TOTAL	41	20	160	80	13	27	35	73	4	7	52	93

APPENDIX

TABLE A

YEAR OF BIRTH

Number of lung cancer cases by year of birth cohort and number and percentage of total included in the case-control study.

Year of Birth	Excluded	Included	Total	% Included
< 1900	19	21	40	53
1900 - 04	8	29	37	78
1905 - 09	11	28	39	72
1910 - 14	4	27	31	87
1915 - 19	4	10	14	71
1920 +	0	4	4	100
	46	119	165	72

TABLE B

YEAR OF DEATH

Number of lung cancer cases in cohorts by year of death and number and percentage included in the case-control study.

Year of Death	Excluded	Included	Total	% Included
1955 - 59	8	5	13	38
1960 - 64	10	13	23	57
1965 - 69	12	37	49	76
1970 +	16	64	80	80
	46	119	165	72

TABLE C

YEAR STARTED GOLD MINING

Number of lung cancer cases in cohorts by year of start of gold mining and number and percentage included in the case-control study.

Year of Start	Excluded	Included	Total	% Included
< 1925	9	13	22	59
1925 - 29	9	28	37	76
1930 - 34	5	25	30	83
1935 - 39	14	15	29	52
1940 -44	4	21	25	84
1945 +	5	17	22	77
	46	119	165	72

TABLE D

AGE AT DEATH

Number of lung cancer cases in cohorts by age at death and number and percentage included in the case-control study.

Age at Death	Excluded	Included	Total	% Included
< 50	4	3	7	43
50 - 54	3	13	16	81
55 - 59	8	26	34	76
60 - 64	9	27	36	75
65 - 69	12	24	36	67
70 +	10	26	36	72
	46	119	165	72

TABLE E

X-RAY RATING

Number of lung cancer deaths included in the case-control study in sub-cohorts by X-Ray rating.

Sub-cohort*	Excluded	Included	Total	% Included
A	35	78	113	69
B + C	11	41	52	79
	46	119	165	72

*Sub-cohort
A: Ontario X-Ray rating less than 4 ILO rating 0/0
B: Ontario X-Ray rating 4 ILO rating 0/1 or 1/0.
C: Ontario X-Ray rating greater than 4 ILO rating 1/1 or greater.

TABLE F

CLASSIFICATION OF EXPOSURE TO BLASTING FUMES BY CALENDAR PERIOD AND TYPE OF UNDERGROUND WORK (FROM HIGHEST EXPOSURE I TO LOWEST EXPOSURE IV.)

Type of work	before 1927	1927-1939	1940-1947	1948-1977
Stope	I	II	III	IV
Raise	I	II	III	IV
Tramming	I	II	III	IV
Drift	I	I	IV	IV
Other	I	III	IV	IV

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Commission des accidents du travail de l'Ontario

Commission de contrôle de l'énergie atomique du Canada

ÉTUDE SUR LA MORTALITÉ DES OUVRIERS DES MINES D'OR DE L'ONTARIO 1955-1977

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RÉSUMÉ

Une étude précédente a montré que les risques de cancer du poumon étaient significativement plus élevés chez les ouvriers des mines d'or de l'Ontario que dans le groupe correspondant de la population masculine de l'Ontario et que chez les ouvriers des mines de nickel et de cuivre. Dans ce rapport, nous examinerons les causes susceptibles d'expliquer ce phénomène.

L'objectif premier de cette étude était d'établir si les risques de cancer du poumon, plus élevés chez les ouvriers des mines d'or, étaient liés à leur occupation ou plutôt à un mode de vie particulier.

Le second objectif visait à rechercher les corrélations possibles entre le cancer du poumon et certains polluants présents dans les mines.

Pour les besoins de cette étude, nous avons effectué une enquête prospective portant sur une période donnée et comprenant en elle-même une enquête rétrospective.

Les conditions d'appartenance au groupe des ouvriers des mines d'or ont été modifiées depuis le dernier rapport. Ce groupe ne comprend maintenant que les mineurs en activité entre le 1er janvier 1955 et le 31 décembre 1977. On a estimé que les ouvriers qui ne travaillaient plus dans une mine d'or de l'Ontario au 1er janvier 1955, mais qui continuaient à subir volontairement des examens prévus pour les mineurs, formaient un groupe distinct. Le fait qu'ils puissent subir ces examens pour des raisons de santé risquait de fausser la suite de l'évaluation. On a donc exclu ce groupe de mineurs des sujets à l'étude. Cette modification a entraîné une réduction du

nombre des décès constatés et prévus imputables à diverses causes, mais n'a pas sensiblement influé sur le risque relatif, à l'exception notable de la silico-tuberculose. Le nombre de décès constatés et attribuables à cette maladie est passé de 41 à 23 entre l'étude précédente et le présent rapport. De même, avec l'introduction d'une nouvelle définition des mineurs, le nombre de décès provoqués par la silicose a diminué, passant de 111 à 39.

Comme on s'y attendait, le tabac était la cause principale du cancer du poumon chez les ouvriers des mines d'or. Chez les fumeurs, le risque relatif est sept fois plus élevé que chez les non fumeurs. Cependant, l'analyse comparative de la consommation de tabac des ouvriers des mines d'or et de ceux des mines de nickel et de cuivre n'a pas révélé de différences importantes susceptibles d'expliquer l'existence de risques plus élevés chez les premiers. De même, il n'y a pas de différences évidentes dans le mode de vie ou les conditions socio-économiques qui puissent expliquer cet écart entre les deux groupes étudiés.

On ne peut pas non plus expliquer les variations qui existent par les différences d'âge entre les ouvriers des mines d'or et ceux des mines de nickel et de cuivre. Les risques sont plus élevés chez les ouvriers des mines d'or à tous les groupes d'âge généralement menacés par le cancer du poumon. L'augmentation la plus sensible se retrouve d'ailleurs chez les 55-64 ans, qui représentent une bonne proportion des ouvriers des mines d'or, des mines de nickel et de cuivre.

Comme nous n'avons pas pu établir que les facteurs relatifs au mode de vie pouvaient offrir une explication acceptable de l'augmentation des risques de cancer du poumon, nous nous sommes alors penchés sur les liens avec le milieu du travail.

On a remarqué que les risques de cancer du poumon chez les ouvriers des mines d'or n'étaient pas liés au nombre total d'années d'exposition en milieu souterrain. Lorsqu'on étudie le rapport entre l'exposition et la réaction, on s'aperçoit que le nombre total d'années de travail sous terre n'est pas nécessairement une mesure adéquate de l'exposition, surtout lorsqu'il existe de grandes variations du taux d'exposition au cours du temps.

Les systèmes de ventilation et de réduction des poussières utilisés dans les mines ont été améliorés au fil des ans, en particulier au milieu des années 1940. De ce fait, nous avons examiné le rapport qui pouvait exister entre le nombre d'années de travail sous terre avant 1945 et les risques de cancer du poumon. Nous avons constaté que ces risques ont sensiblement augmenté avec la durée d'exposition aux poussières des mines avant 1945, mais qu'après cette date, l'exposition n'a joué qu'un rôle minime.

Le lien entre le début de l'exploitation des mines d'or et les risques de cancer de poumon a été prouvé. Les hommes qui ont commencé à travailler il y a très longtemps dans les mines d'or, alors que les conditions étaient moins favorables, sont plus menacés que ceux qui ont commencé à travailler à une date ultérieure.

Les résultats de l'étude rétrospective montrent que les risques de cancer du poumon augmentent en fonction du nombre de mois passés dans un milieu à forte exposition aux poussières.

Tous les résultats mentionnés jusqu'à présent montrent que le métier des ouvriers des mines d'or contribue significativement à accroître les risques de cancer du poumon.

Le second objectif de l'étude visait à établir si les risques de cancer du poumon pouvaient être liés à l'exposition à certains polluants présents dans les mines d'or. Parmi les

facteurs étudiés, citons la silicose, l'exposition à des taux élevés de poussière, l'exposition à la silice, à l'arsenic, aux matières fibreuses présentes dans le minerai et la radioactivité.

La silicose nous est apparue particulièrement suspecte. Des études expérimentales ont prouvées que l'inhalation de la silice pouvait provoquer, dans certaines circonstances, le cancer du poumon chez les rats, observations également confirmées par certains travaux épidémiologiques.

Les données de cette étude montrent une augmentation importante des risques de cancer du poumon dans tous les sous-groupes de mineurs non silicotiques et silicotiques. Le risque relatif de mourir d'un cancer du poumon croît cependant des non silicotiques aux silicotiques selon la catégorie de classification des radiographies (sous-groupes A à C).

Lorsqu'on a comparé la consommation de tabac des mineurs silicotiques et non silicotiques, on a remarqué que les premiers fumaient plus que les seconds. Cette constatation vient confirmer l'hypothèse selon laquelle le tabac augmente les risques de silicose dans les milieux où elle est susceptible de se produire, ce qui entraîne indirectement une augmentation des risques de cancer du poumon chez les silicotiques.

Le rapport entre d'une part, le tabac et la silicose et d'autre part, les risques de cancer du poumon a fait l'objet d'une analyse à variables multiples. On a pu établir que chacun des deux facteurs, le tabac et la silicose, était étroitement lié aux risques de cancer du poumon. Par ailleurs, l'association avec la catégorie de la classification des radiographies ne pouvait plus être prouvée lorsque le tabac était inclus dans le modèle.

Ces résultats confirment que le tabac augmente les risques de cancer du poumon et de silicose et que la plus grande vulnérabilité des silicotiques par rapport aux non silicotiques provient au moins en partie du degré de consommation de tabac des différents sous-groupes.

On peut également penser que la silicose dont sont atteints les ouvriers des mines d'or de l'Ontario est le signe d'une forte exposition aux poussières, qui entraîne une exposition aux agents cancérogènes présents dans les mines, ce qui laisserait alors penser que le rapport entre la silicose et l'augmentation des risques de cancer du poumon est probablement erroné.

Les données géologiques sur les différentes mines d'or ont été fournies par la Commission géologique de l'Ontario. Les mines ont été reparties en plusieurs groupes selon les facteurs suivants :

taux élevé ou faible d'arsenic
taux élevé ou faible de matières fibreuses
taux élevé ou faible de sulfure
intensité forte ou faible de radioactivité
taux élevé ou faible de silice

On a utilisé plusieurs modèles de risque relatif pour étudier le rapport entre les risques de cancer du poumon et les paramètres ci-dessus d'une part, et les données sur l'exposition aux poussières et le tabac d'autre part. L'application de ces modèles aux ouvriers des mines d'or de l'Ontario semble indiquer un rapport étroit entre les risques de cancer du poumon et l'exposition combinée à des taux élevés de poussières, d'arsenic et/ou de matières fibreuses. La corrélation avec l'exposition à la silice ou à des taux élevés de radon, déduits de la géologie, n'a pas pu être prouvée. On suppose donc que la présence de taux élevés d'arsenic et de

matières fibreuses dans le minerai est liée à des risques plus importants de cancer du poumon. Il semble également que les taux élevés de poussières, combinés à des taux considérables d'arsenic et de fibres, ne soient pas les seuls agents cancérogènes dans les mines. En effet, les données de l'étude prospective indiquent une sensible augmentation des risques de cancer du poumon même chez les mineurs qui n'ont jamais travaillé dans des milieux caractérisés par des taux élevés de poussières, d'arsenic et de matières fibreuses. L'influence d'autres facteurs propres à l'environnement minier, comme le radon et ses dérivés à courte période radioactive, n'est donc à nier.

Il serait bon de souligner cependant que la géologie ne permet pas de déterminer avec exactitude les taux élevés des dérivés du radon dans les mines. Or, on ne pouvait pas utiliser de moyen plus direct pour examiner le rapport possible entre l'exposition au radon et ses dérivés dans l'atmosphère des mines et les risques de cancer du poumon. On ne connaît pas les taux antérieurs des dérivés du radon dans les mines d'or de l'Ontario et les résultats des évaluations récentes ne sont disponibles que dans certaines des mines où travaillaient certains sujets du groupe étudié.

Les conséquences possibles de la prévention de la silicose par l'aluminium sur les risques de cancer du poumon ont également été étudiées. Sur les 7057 sujets qui constituent le groupe des ouvriers des mines d'or, seuls 327 mineurs (soit cinq pour cent) ont déclaré ne pas avoir été exposés à cette méthode. L'analyse des deux groupes, mineurs exposés et non exposés, révèle dans les deux cas des risques de cancer du poumon nettement plus importants, mais sans différence notable entre les deux groupes. Les risques de cancer du poumon sont plus élevés chez les ouvriers qui ont travaillé dans les mines d'or de l'Ontario avant 1936, ou dix ans environ avant l'introduction de la méthode de prévention par l'aluminium, que chez les mineurs qui ont commencé à travailler à une date

ultérieure. L'étude rétrospective, quant à elle, n'indique aucun rapport entre les années d'exposition à l'aluminum et les risques de cancer du poumon. L'hypothèse d'une augmentation des risques de cancer du poumon chez les ouvriers des mines d'or de l'Ontario, liée à l'utilisation de la méthode de la prévention de la silicose par l'aluminium, n'a donc pas été vérifiée par les données de cette étude.

La comparaison de la classification des cancers du poumon par type de cellule, effectuée dans les hôpitaux, et de la nouvelle classification du docteur Ritchie a révélé des divergences fondamentales, que nous aborderons dans une autre publication. Pour les besoins de cette étude, nous nous sommes limités à l'examen des résultats de la nouvelle classification. Selon cette dernière, aucune différence importante dans la répartition des cancers du poumon par type de cellule n'a été observée entre les ouvriers des mines d'or, de nickel et de cuivre.

Aucun agent directement responsable de l'augmentation du taux de cancers de l'estomac n'a été défini. Par ailleurs, il a été prouvé qu'il n'y avait aucun rapport entre la forte proportion d'immigrants dans le groupe étudié et les taux élevés de cancer observés.

L'objet de cette étude était d'établir si les taux de cancer plus élevés que l'on a constatés chez les ouvriers des mines d'or par rapport à la population masculine de l'Ontario et aux ouvriers des mines de nickel et de cuivre était liés à des modes de vie ou à des environnements miniers différents. Nous cherchions à prouver que l'augmentation des risques provenait de l'exposition à certains éléments du milieu minier, nous nous serions alors efforcés de définir le ou les agents cancérogènes.

Il s'est avéré nécessaire de modifier la définition des ouvriers des mines d'or en milieu souterrain, que nous avions utilisée dans le rapport de 1983 mais qui avait faussé la situation, ce qui a entraîné, pour le groupe en question, une réduction importante du nombre des décès attribuables à la silico-tuberculose et à la silicose. Les risques relatifs de décès imputables à d'autres causes, notamment le cancer du poumon, demeurent pratiquement inchangés.

CONCLUSIONS :

1) Il est fort peu probable que l'augmentation des risques de cancer du poumon chez les ouvriers des mines d'or de l'Ontario provienne de modes de vie différents, notamment le facteur tabac, entre les ouvriers des mines d'or, des mines de nickel et de cuivre et le groupe correspondant de la population masculine de l'Ontario.

2) Les données indiquent un rapport direct entre le travail sous-terre et l'augmentation des taux de cancer du poumon chez les ouvriers des mines d'or.

3) Le début de l'exploitation souterraine des mines d'or de l'Ontario et la durée d'exposition aux poussières dans ces mines avant 1945 sont liés à l'augmentation des risques de cancer du poumon. Après 1945, l'exposition devient un facteur insignifiant.

4) Les données révèlent également un rapport évident et important entre la catégorie de classification des radiographies et les risques de cancer du poumon, lien qui disparaît en grande partie lorsqu'on tient compte des différences qui existent entre les mineurs silicotiques et non silicotiques au niveau de la consommation de tabac. Dans un milieu où la silicose est susceptible de se produire, le tabac augmente les risques de silicose et de cancer du poumon.

5) Chez les ouvriers des mines d'or, la silicose n'est pas seulement liée au tabac mais le signe également d'une forte exposition aux poussières contenant de la silice libre et tout autre agent cancérogène.

6) Le lien entre les risques de cancer du poumon et l'exposition à des taux élevés de poussières comprenant une quantité importante d'arsenic demeure faible.

7) Le rapport entre les risques de cancer du poumon chez les mineurs et l'exposition aux dérivés à courte période radioactive du radon 222 n'a pas pu être examiné car nous ne disposons pas de données antérieures sur les taux des dérivés du radon dans les mines d'or. Aucun rapport entre les risques du cancer du poumon et la radioactivité dans les mines, déduite des données géologiques, n'a pu être établi.

8) Aucun lien entre les risques de cancer du poumon chez les ouvriers des mines d'or et l'exposition à l'aluminium (méthode de prévention de la silicose) n'a pu être établi.

9) La répartition des cancers du poumon par type de cellule ne révèle aucune différence significative entre les ouvriers des mines d'or, de nickel et de cuivre .

10) Aucun agent cancérogène directement responsable de l'augmentation du taux de cancer de l'estomac chez les ouvriers des mines d'or n'a été défini.